APPENDIX.

TABLE OF MAJOR OPERATIONS ON THE LONG BONES.

Kashmir Mission Hospital, January 1, 1882, to December 31, 1886.

Operation.	Femur.	Tibia.	Fibula.	Humerus.	Radius.	Ulna.
Subperiosteal resection, primary or secondary	2	10	4	2	2	3
Chiselling or gouging	10	21	1	3	1	...
Sequestrotomy	20	21	...	3	2	1
Bone drainage by osteotomy, etc.	...	12	...	...	...	...
Total	32	64	5	8	5	4

This list does not include the very numerous cases of periosteotomy, opening abscesses, etc. Of the whole, only two cases (mentioned in the text) were fatal. None were amputated. All regained useful limbs.

CASE OF BILATERAL OPHTHALMOPLEGIA EXTERNA AND INTERNA ASSOCIATED WITH TABES DORSALIS, BULBAR PARALYSIS, AND LOSS OF VISION AND HEARING.

WITH SOME OBSERVATIONS UPON THE PATHOGENY AND ETIOLOGY OF BILATERAL OPHTHALMOPLEGIA EXTERNA.[1]

By R. P. Howard, M.D., LL.D.,
PROFESSOR OF MEDICINE IN MCGILL UNIVERSITY, MONTREAL.

ALTHOUGH von Gräfe, in 1856,[2] described "progressive ophthalmoplegia," it was not until 1879 that Jonathan Hutchinson aroused the attention of English physicians to that affection under the title "Ophthalmoplegia Externa,"[3] or paralysis of the external muscles of the eyeball. In the preceding year he had employed the term "ophthalmoplegia interna"[4] to indicate loss of power of the internal musculature of the eye, the iris and ciliary muscle. The affection described by the eminent English surgeon, however, was not the very common one in which the external muscles supplied by the third or the sixth nerve of one eye are paralyzed, partial oculo-motor paralysis, but that in which there is bilateral or symmetrical immobility of both eyes with ptosis.

Now, clinically, it is very important to distinguish between unilateral ophthalmoplegia and the symmetrical bilateral form as the causes or conditions of occurrence are not the same in both. The bilateral, if involving all the external muscles of the eyes, but exempting the internal, very frequently, but not always, indicates disease of the nuclei situated in the floor of the fourth ventricle and of the aqueduct of Sylvius from which the oculo-motor nerves arise. It is often associated with similar degeneration in the nuclear origins of other cerebral or spinal nerves, as in bulbar paralysis and progressive muscular atrophy, or in the posterior columns of the cord as in tabes dorsalis.

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[1] A paper read at the meeting of the Canada Medical Association, 1888.

[2] Arch. f. Ophth., Bd. ii, p. 293, 1856.

[3] Med.-Chirurg. Trans., lxii. p. 308.

[4] Ibid., lxi. p. 215.

Through the kindness of several colleagues, I had occasion to see the following case in consultation September 9, 1884:

Patient, an educated man fifty-four years of age, was of studious and very temperate habits, and free from evidences of syphilis, the possibility of which disease he positively denied. He married at thirty-three, and was the father of sixteen children; the youngest eight months old. About the beginning of August, 1883, suffered from severe headache, which he regarded as migraine, having been much afflicted with that neurosis in his youth; at the same time he became irascible. Toward the end of the month strabismus, diplopia, and slight ptosis on the left side supervened, but these gradually disappeared in about six weeks. Some five months later, lateral deviation of the right eye with diplopia set in, and in about two months was followed by ptosis, which like the other symptoms developed gradually, but unlike them has proved permanent.

In January, 1884, vision began to fail, and very soon reading produced ocular fatigue and frontal headache. In March articulation gradually grew difficult and slow—he had been a rapid speaker. He experienced no difficulty in selecting words to convey ideas, no failure of memory or intelligence. In April power of mastication became enfeebled, so that finally the lower jaw dropped from the upper and he could not raise it without the aid of his fingers. Deglutition likewise suffered; solids were occasionally arrested and ejected; fluids, including the saliva, trickled from the open mouth. In March his appetite left him, he became very weak and despondent, steadily lost flesh, and took to his bed from general debility. At this time his sexual power declined rapidly. In July deafness set in gradually and at first intermittingly. In the early part of August vision of the right eye failed rapidly. Of late he has been able to elevate the right eyelid a little and has regained somewhat masticatory power. Since March he has had several syncopal attacks, in which, although he does not lose consciousness, he grows very faint, vision fails, and his pulse becomes scarcely perceptible.

Present state.—Pale and much emaciated; surface dry and polished looking, voice feeble. Relates his own history, but so indistinct and defective is his articulation that I cannot understand many of his words. Marked ptosis on right side; can slowly raise the lid sufficiently to expose lower half of pupil; slight ptosis on left side; eyeballs fixed, no lateral or vertical movements; axes normal in direction; no strabismus; right pupil moderately large; left of medium size; both unaffected by light, and in both power of accommodation lost; complete blindness of right eye; reads with difficulty medium print with left eye—but two or three words fatigue him; eyes cannot follow an object passed before them; has occasional darting transitory pains in the eyeballs, unaccompanied by luminous phenomena.

Right side of face smoother than left and angle of mouth drawn slightly to the left; cannot whistle; can contract orbicularis palpebrarum well, but the vertical folds are not as pronounced in the right upper lid as in the left. Lower jaw at rest is pendant; when speaking he closes it; asked to close the jaws, he does so by lifting his chin with two fingers; says that he has more power of mastication than he had, yet he cannot bite my finger placed between his teeth; little if any lateral movements of inferior maxilla; the masseter and temporal muscles on both sides are wasted, and can hardly be felt to contract during mastication; tongue when protruded deviates to the right, and its right half appears to be slightly atrophied.

No loss of power beyond general weakness in arms and hands; grasp of dynamometer by right hand 82, by left 78; in the horizontal posture can raise either leg when extended in spite of considerable pressure applied in front of ankle.

Tick of watch not heard when watch is applied either to ear or mastoid region; at speaking distance a moderately loud voice is heard; smell and taste unimpaired; function of bladder fairly performed; habitual constipation; appetite and digestive power now good; lives chiefly upon milk and soup; pulse 78, not strong; systolic basic murmur over heart.

Sept. 13. Reëxamined patient and confirmed the above facts, and learned some additional ones. Tactile and thermal sensibility lost over right frontal region, especially over supra-orbital region, less impaired over the infra-orbital area; not impaired over mental area; sensation on left side of face normal except in the temporal and supra-orbital regions; cornea of either eye insensitive to touch, and no reflex contraction of lid produced thereby; sensibility of both sides of tongue and of soft palate normal; no numbness in soles of feet; absence of patellar-tendon reflex on either side; prompt contraction of vastus internus under percussion of finger; walks readily without any ataxia with eyes shut, but totters from debility; has had for some time sense of stiffness of right cheek, and the finger introduced into mouth finds that the right cheek is forcibly applied to the teeth; at my request an oculist examined the retina, and on the 20th he reported the existence of that form of optic atrophy in either eye that occurs in locomotor ataxia, and loss of vision.

Another opportunity of seeing the patient was not afforded me, and he died two months later. There was no autopsy.

Here, then, we have a definite, yet progressive, invasion in the nervous system in a previously healthy and temperate man, free from syphilis, the steps, so to speak, of which were: headache, irascibility, diplopia, strabismus, and slight ptosis on the left side, which disappeared gradually. Five months later a gradual but permanent establishment of the same symptoms on the right side. A few months later gradual impairment of articulation, mastication, and deglutition, and about the same time the loss of sexual power. Four months later, intermitting and then continuous progressive bilateral impairment of hearing; a month later, rapid failure of vision in right eye, and soon thereafter a tendency to syncopal seizures.

The order of development of the following additional symptoms present when he first came under my observation cannot be stated, as they had not been investigated: loss of pupil reflexes and of accommodation; complete immobility of both eyes, and occasional transitory darting pains in them; loss of the patellar-tendon reflex; insensitiveness of both corneæ, with absence of corneal reflex; loss or impairment of tactile and thermal sensibility in same branches of both fifth nerves—paresis of associated muscles—labial (supplied by the facial nerve) and lingual (supplied by the hypoglossal).

Resolving the symptoms present in this case into the several groupings which they appear to form, we have chronic progressive ophthalmoplegia externa and interna, chronic labio-glossal paralysis, and tabes dorsalis, combined with bilateral motor paralysis of the fifth nerve; loss of two of the special senses, vision and audition; and impairment of common sensation in some of the branches of the sensory portion of the fifth nerve on both sides.

The direct evidence of the existence of tabes dorsalis is the absence of the patellar-tendon reflex and the loss of sexual power without paralysis of the lower extremities; and the indirect, is the association with the above of double optic atrophy and amaurosis; early disturbance of the external and internal ocular muscles.

It is to be regretted that an examination of the larynx was not made, nor the degree of mobility of the soft palate recorded; but the coexistence of the following symptoms: great impairment of articulation and deglutition, inability to whistle, paralysis of the tongue, with slight wasting of one side of it, points to bulbar paralysis. Had the patient lived longer, paralysis, I doubt not, and wasting of both sides of the tongue would have been more marked.

It is not necessary to go over the evidence of the presence of progressive bilateral ophthalmoplegia. I have already alluded to the frequent association of chronic symmetrical bilateral ophthalmoplegia with other chronic affections of the nervous system, especially tabes dorsalis, and bulbar paralysis, and its occasional coexistence with progressive muscular atrophy and with "general paresis."

PATHOGENY.—As these chronic associates of progressive symmetrical ophthalmoplegia present as their essential lesion a degeneration, consisting of an atrophy of the nerve elements and hypertrophy of the connective tissue of the affected portion of the columns, horns, bulb, or cortex, so in a few instances of progressive ophthalmoplegia that have been examined carefully[1] after death, an identical degeneration of the elements of the central nuclei, from which the oculo-motor nerves arise, has been found, hence the name recently applied to these cases, "Nuclear Paralysis."

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[1] Med.-Chir. Trans., lxii. p. 316; Gower's Dis. Nerv. System, ii. p. 185.

Chronic progressive ophthalmoplegia belongs, as a very general rule, to the "system diseases" of the cerebro-spinal system, depending, as it does, upon the morbid conditions of structures which have a common function, the regulation of the movements of the external and internal muscles of the eyes. Participation in function and secondary degeneration of tracts or centres connected with the primarily diseased area are clews to the pathogeny of bilateral ophthalmoplegia; but they do not explain the coexistence of bulbar paralysis or of tabes dorsalis, etc., sometimes present with it. Why, in such a case, does a distinct and different "system disease," a labio-glossal paralysis, or a posterior spinal sclerosis, or, as in our case, *both of these*, become associated with bilateral ophthalmoplegia externa? When the close proximity of the motor and even of the sensory nuclei of the cerebral nerves is recalled,[1] simple *extension* of disease by contiguity will doubtless be a part of the answer to this question.

Disease of the bloodvessels supplying the centres, such as endo- and peri-arteritis and senile atheroma, may also partly account for degeneration and other lesions of different centres or tracts which receive their blood supply from the same vessels.[2] Senile atheroma especially is apt to be diffused over the general arterial system, and the consequent atrophy or sclerosis may then invade indifferently and at the same time or successively, functionally related "systems" and structures of distinct and different functions.

Syphilis would seem to exercise some not well understood influence in predisposing to, if not inviting, degenerative processes in the oculo-motor nuclei as well as in the posterior spinal column, and this may account for the coexistence of ophthalmoplegia externa and locomotor ataxia, or of ophthalmoplegia and general paralysis of the insane. Syphilis is especially prone to produce endo- and peri-arteritis, and this is doubtless one way in which it imitates a diffused sclerosis or degeneration in the brain or cord. Finally, some congenital defect, whether an inherent want of resisting power to morbid influences, or an imperfect development of certain tracts, may account for the facility with which degeneration or other morbid process affects different portions of the cord or brain.[3]

As regards the *etiology* of bilateral ophthalmoplegia, the *acute* form has been met with in *apoplexy*, the blood occupying the region of the oculo-motor nuclei[1] or pressing upon the corpora quadrigemina and floor of the third ventricle, in a case of basic tuberculous meningitis,[2] as a rare sequence of diphtheria[3] and of typhus fever,[4] in a case of sulphuric acid poisoning, and in three of chronic alcoholism.[5]

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[1] Exhibited diagrams of nuclear origin of cerebral nerves.

[2] See Adamkiewitz: Trans. International Med. Cong., London, pp. 1-3, 1881.

[3] Vide Ormerod, Brain, 1884; Bury, Ib., 1886; Smith, Boston Med. and Surg. Journal, 1885.

When alcoholism or diphtheria induces ophthalmoplegia, it is probably sometimes through a *peripheral neuritis.* In Suckling's[6] case, due to alcohol, there were symptoms of peripheral neuritis in the lower extremities associated with the ophthalmoplegia. In the diphtheritic form it is probably rather a simple degeneration of the nerve fibre than a real neuritis, the interstitial tissue of the nerves very seldom exhibiting the proofs of inflammation except in the nerves of the palate.[7] The degeneration may affect the *periphery* of the nerves in their *whole extent,* including their anterior roots.

Diphtheritic ophthalmoplegia is probably sometimes of *nuclear* origin; just as diphtheritic paralysis of the spinal nerves is, in severe cases, sometimes associated with alteration of their ganglion cells in the cord. This view is favored by the fact that capillary hemorrhages have been found in the nerve centres and nerves in diphtheria. Professor W. M. Thomson,[8] however, has quite lately objected to the doctrine that diphtheritic paralysis depends upon lesions of the nervous centres, and has advocated its peripheral origin. The weight of evidence, in my opinion, supports the statement, that while diphtheritic paralysis is most frequently caused by peripheral neuritis, it is occasionally an effect of lesion of the nerve centres.

Finally, acute ophthalmoplegia has been observed in connection with bulbar paralysis and with facial palsy combined with paraplegia.

Chronic bilateral ophthalmoplegia, *per se,* is most frequently found in syphilis[9] affecting the oculo-motor nerves from their points of emergence from the base of the brain to the cavernous sinus, or in the walls of that sinus; or possibly affecting their nuclei directly or probably indirectly through disease of the vessels which supply these nuclei. It is also frequently observed in association with tabes dorsalis and occasionally in combination with progressive muscular atrophy, chronic bulbar paralysis and "general paresis;" and is then mostly due to nuclear degeneration, these associated diseases severally depending upon degeneration of the nerve fibres and cells of the cord or the brain.

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[1] Sturge: Brit. Med. Journal, i. p. 851, 1881; Thomas: Ib., i. p 763, 1882.

[2] Mules: Brit. Med. Journal, xi p. 127, 1887.

[3] Gowers, Lib. cit., cites two cases: Uhthoff Neur. Centralbt., 1885, and Mendel, Ib., corœnne Bull· de l'Soc. Franc. d'Oph.

[4] Corœnne, Ib.

[5] Gowers, Lib. cit., xi. p. 182, from Gehirn Krankheiten, xi. p. 233.

[6] Suckling, Brit. Med. Journal, i. p. 464, 1888.

[7] Gowers, Lib. cit., ii. p. 838.

[8] The Med News, Philadelphia, June 9, 1888.

[9] Hutchinson: Med.-Chir. Trans., lxii.; Seguin; Journ. Nervous and Mental Dis., 1888, 317, and many others.

Thrombosis and inflammation of the cavernous sinuses are occasional causes of more or less complete progressive bilateral ophthalmoplegia.[1] It is then usually at first unilateral, but as the thrombosis is very prone to extend from one cavernous canal to the other,[2] the paralysis often becomes bilateral, and sometimes the muscles of the eye primarily affected regain their power. In some instances, internal ophthalmoplegia is joined to the external form (Robinson and Hutchinson's case). This implication first of one eye, then of the other, was regarded by Dr. Coupland as pathognomonic of ophthalmoplegia caused by thrombosis of the cavernous sinuses, but in the case which forms the subject of this paper there were no grounds for suspecting disease of the sinus, yet the ophthalmoplegia was unilateral for five months before it invaded the other side.

Tubercle at the top of the pons near the aqueduct of Sylvius was supposed to have been the causative lesion in Berry and Bramwell's case observed in a child two and a half years old.[3] The patient recovered, hence no opportunity was afforded of testing the accuracy of the diagnosis. In a case presenting almost the same clinical features as the last, Kojewnikoff[4] found capillary hemorrhage with softening of the gray matter limited to the floor of the fourth ventricle, the confines of the nucleus of the third nerve and aqueduct of Sylvius, extending symmetrically upon the two sides. Softening from embolism or thrombosis of the vessels of the floor of the fourth and third ventricles may be a rare cause of the disease.[5] Cold has been the only apparent cause in a few cases, and so have blows on the head, probably through basilar meningitis, or hemorrhage, or basilar periostitis being thereby reproduced.

Cases are published in which bilateral ophthalmoplegia externa has been a congenital and inherited defect. In some of these the ocular muscles and not their nerves seemed to be the source of the affection, for in a few instances the muscles have been found absent, in others they have been shorter and their sclerotic insertions placed more backward than what is normal. W. Lawford met with three instances of this inherited form in a family of seven children.[6]

In some cases observed in early life and apparently due to muscular defects, the primary disease may have been in the nerve centres and have been followed by atrophy or arrested development of the ocular muscles.

Well-marked cases of bilateral ophthalmoplegia externa uncomplicated by *other* evidences of disease of the nervous system are recorded[1] in which *no cause* could be assigned. In one of these our young Canadian representative in the metropolis, W. Lawford, "thought the nuclei involved were those controlling the bilateral coördinated movements excited through the third, fourth, and sixth nerves, rather than the nuclei of origin of those nerves, for the reason that the action of the iris and ciliary muscles remained intact in each eye." But it is now admitted[2] that the nuclei which supply the internal muscles of the eye *lie in front* of those which supply the external muscles, and they are not supplied by the same vessels, so that the nuclei of the external ocular muscles may suffer and the nuclei of the internal escape, and *vice versa.*

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[1] Coupland: Trans. Ophthal. Soc. of United Kingdom, 1887, vii. 223; Robinson and Hutchinson, Jr., Ib., 230, with plate.

[2] Thirteen times in twenty-two cases collected by Coupland.

[3] Edinburgh Med. Journ., 1857.

[4] Annual of the Universal Med. Sciences, 1888, iii. 143, quoted from Prog. Médicale.

[5] Kohler and Peck, Zeitschr f. Heilk., Prag., iii. 1880-81.

[6] Brit. Med. Journ., ii., 1887.

It is perhaps possible that a *tumor* compressing the floor of the fourth ventricle and Sylvian aqueduct may cause bilateral ophthalmoplegia externa, but it must be an exceedingly rare occurrence and there would in all probability coexist other evidences of cerebral tumor, the most reliable perhaps of which would be double optic atrophy—not referable to tabes.

In addition to these causes which are severally productive of bilateral external ophthalmoplegia through a *gross lesion* of the oculo-motor centres or nerves, recent clinical experience has brought to light cases of the disease apparently of *functional* origin, and belonging to the neuroses, as idiopathic epilepsy and hysteria, etc., do.

A very interesting case of ophthalmoplegia externa associated with Graves's disease was published by Dr. Francis Warner[3] in 1882, and its sequel is related by Dr. Bristowe[4] in an able paper contributed in 1885, under the title "Cases of Ophthalmoplegia, complicated with various other Affections of the Nervous System." In addition to Graves's disease, this patient became the subject of bilateral ophthalmoplegia externa, gastric crises, complete right hemianæsthesia, great nervousness, constantly recurring hemorrhages from the ears and right nostril, and subsequently epileptic fits, persistent right hemiplegia with rigidity, and an almost constant temperature ranging from 101° to 105°. She died of bronchitis. A careful microscopical examination failed to detect "even a trace of disease" in the brain, cord, or intra-cranial tissues.

Bristowe's second case was almost a counterpart of his first, but the right-sided hemianæsthesia was not complete, the cerebral hemorrhage and Graves's disease were absent, and a left-sided chorea formed an episode of the case: this girl gradually recovered from her ophthalmoplegia, right hemiplegia, epileptic fits, etc.

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[1] J. B. Lawford: Trans. Opth. Soc. of United Kingdom, vii. 260, and Brit. Med. Journ., 1887, ii. p. 127; Belvor: Trans. Opth. Soc., vii. p. 281, and Brit. Med. Journ., 1887, i. p. 622; Hutchinson, Ib., 1888, i. p. 137; Birdsall: Journal Nervous and Mental Dis., 1887, p 66.

[2] Hensen and Völckers, Kohler and Pick, Jairhand. Forster, Graves.

[3] Med.-Chir. Trans., vol 66. pp. 107, 112.

[4] Brain, Oct. 1885, pp. 313, 314; Ib., Jan. 1887, pp. 544, 546.

Dr. Gilbert Ballet[1] last May contributed a personal observation of a case of exophthalmic goitre associated with paralysis of the ocular and facial muscles, a general anæsthesia, *i. e.*, loss of all special and common sensation, the loss being most profound on the left half of the body—bilateral ophthalmoplegia externa—diabetes insipidus, hysteria, and slight impairment of tongue movements. He *quotes* also a case[2] in which external ophthalmoplegia was combined with Graves's disease, paralysis of the facial, the motor branch of the fifth, and the hypoglossal; no hysterical symptoms coexisted.

Notwithstanding Dr. Bristowe's opposite opinion, I cannot help coinciding with Dr. Ballet in the view that the two patients of the former were the subjects of that functional disturbance we call hysteria, and that the bilateral paralysis of the ocular muscles was of that nature. The association of the ophthalmoplegia with Graves's disease in two of these cases is not opposed to this view, for the latter disease itself belongs to the neuroses or functional affections of the nervous system, and is not infrequently coexistent with hysteria. The important fact is established then, that bilateral ophthalmoplegia externa, although usually an evidence of *organic* disease of the oculo-motor nuclei or nerves, is sometimes a *functional* affection and then is a more hopeful and sometimes a curable affection. The functional variety is of either nuclear origin or more probably depends upon disturbance of oculo-motor centres higher up in the cerebral organism, those perhaps which have to do with volitional motor centres, in the cortex angular gyrus.

It is interesting, in this connection, to note that in these four cases of functional ophthalmoplegia externa, while the external or *voluntary* muscles of the eyes were paralyzed, the internal or *avolitional* muscles were not, the ciliary and the iris muscles continued to perform their reflex and coördinate movements. This is perhaps one very valuable diagnostic difference between functional *bilateral ophthalmoplegia* externa and the more frequent variety due to structural disease.

Amongst many points of interest which this example of bilateral ophthalmoplegia presents, there is one only that I will venture to allude to, as this paper is already too long, viz., the coincidence of the lesion of the motor and sensory roots of the trigeminus which Dr. Seguin,[3] who has also reported an instance, says, on the authority of Dr. Starr, "is almost, if not quite, without precedent."

In conclusion, it may be said that the facts we have mentioned in connection with the etiology of bilateral ophthalmoplegia externa seem to contradict the statement recently made that such paralysis is always due to central or nuclear disease.[1] We have just seen that it may be also of neural or of peripheral origin, as when due to thrombosis and inflammation of the cavernous tissues, to syphilis, diphtheria, and chronic alcoholism, which may produce either inflammation or degeneration of the oculo-motor nerves themselves. A blow on the head must also be recognized as a possible peripheral cause. Further, the affection may be a purely functional one of hysterical nature and the prognosis be then favorable.

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[1] Revue de Méd., May, 1888, pp. 338, 344.

[2] Ibid., p. 363 from Arch. *für* Psychiatrie, B. xvii. Heft. 2, Jandrássik.

[3] Jl. Nervous and Mental Disease, May, 1888, p. 317.

Again, it follows from the facts adduced that bilateral external ophthalmoplegia is frequently at first unilateral and not symmetrical, and it may be impossible to say with certainty whether a unilateral case will become bilateral or not.

Owing to the close proximity of those nuclei of the third pair which preside over the movements of the ciliary muscle and the iris, to those nuclei of the same nerve which regulate the movements of the external muscles of the eyeballs, it frequently happens that bilateral external ophthalmoplegia coexists with bilateral internal ophthalmoplegia; and the circumstance that these nuclei are distinct with functions proper to each accounts for the existence of ophthalmoplegia interna or externa independently each of the other. As a final remark, those interested in the subject of the diagnosis of the several varieties of palsy of the ocular muscles will find a valuable paper by Dr. Allen Starr in the last May number of the *Journal of Nervous and Mental Diseases.* He has there adduced some striking reasons for a new arrangement of the position of the nuclei of the third nerve on the floor of the fourth ventricle differing from both Hensen and Vöelcher's and from Kohler and Pick's.

SUPERNUMERARY MAMMÆ AND NIPPLES IN MAN, MONKEYS, COWS, ETC.,

By J. Bland Sutton, F.R.C.S.,
HUNTERIAN PROFESSOR, ROYAL COLLEGE OF SURGEONS; ASSISTANT SURGEON, MIDDLESEX HOSPITAL.

Much that is fanciful and speculative is mixed with the subject of atavism, and the widespread acceptance of the principles of evolution has had the effect of rendering us less critical in the examination of suspected cases. In this short paper I propose to consider briefly the main facts connected with supernumerary nipples and mammæ, for the purpose of showing the care necessary for the proper discrimination of

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[1] Dr. M. A. Starr: Journal Nervous and Mental Dis., May, 1888, p. 316; and Trans. Ophthal. Soc. United Kingdom, vii.